

WEST Search History

DATE: Friday, August 08, 2003

Set Name Query
side by side

Hit Count Set Name
result set

DB=USPT; PLUR=YES; OP=OR

L5	L3 and (pathogen same susceptibility)	75	L5
L4	L3 and (pathogen same susceptible)	83	L4
L3	L2 and mutat\$3	1784	L3
L2	L1 or C same elegans	6447	L2
L1	nematode	5554	L1

END OF SEARCH HISTORY

FILE 'HOME' ENTERED AT 13:10:11 ON 08 AUG 2003

=> medline biosis embase agricola scisearch caplus

MEDLINE IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.

For a list of commands available to you in the current file, enter

"HELP COMMANDS" at an arrow prompt (=>).

=> file medline biosis embase agricola scisearch caplus

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.21

0.21

FILE 'MEDLINE' ENTERED AT 13:10:37 ON 08 AUG 2003

FILE 'BIOSIS' ENTERED AT 13:10:37 ON 08 AUG 2003

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FILE 'AGRICOLA' ENTERED AT 13:10:37 ON 08 AUG 2003

FILE 'SCISEARCH' ENTERED AT 13:10:37 ON 08 AUG 2003

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FILE 'CAPLUS' ENTERED AT 13:10:37 ON 08 AUG 2003

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=> s nematode

L1 98424 NEMATODE

=> s l1 and mutagen?

L2 690 L1 AND MUTAGEN?

=> s MAPK

L3 37796 MAPK

=> s l3 and esp-2

L4 0 L3 AND ESP-2

=> s l3 and esp-8

L5 1 L3 AND ESP-8

=> s l3 and pmk-1

L6 11 L3 AND PMK-1

=> s l6 and l2

L7 1 L6 AND L2

=> s l5 and l2

L8 1 L5 AND L2

=> dup rem

ENTER L# LIST OR (END):16

PROCESSING COMPLETED FOR L6

L9 5 DUP REM L6 (6 DUPLICATES REMOVED)

=> d l9 tot ibib abs

L9 ANSWER 5 OF 5 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2001652695 MEDLINE
DOCUMENT NUMBER: 21561224 PubMed ID: 11703092
TITLE: Isolation and characterization of **pmk**-(1
-3): three p38 homologs in *Caenorhabditis elegans*.
AUTHOR: Berman K; McKay J; Avery L; Cobb M
CORPORATE SOURCE: Department of Pharmacology, University of Texas
Southwestern Medical Center at Dallas, 5323 Harry Hines
Boulevard, Dallas, TX 75390, USA.
CONTRACT NUMBER: GM 53032 (NIGMS)
HL 46154 (NHLBI)
SOURCE: MOLECULAR CELL BIOLOGY RESEARCH COMMUNICATIONS, (2001 Nov)
4 (6) 337-44.
Journal code: 100889076. ISSN: 1522-4724.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20011114
Last Updated on STN: 20021015
Entered Medline: 20020130

AB p38, a member of the mitogen-activated protein kinase (**MAPK**) superfamily, is activated in response to a variety of cellular stresses and ligands. Since the genome of the nematode *C. elegans* has been sequenced, we sought to identify and characterize the nematode homolog of mammalian p38. By sequence analysis and RT-PCR, we isolated cDNAs encoding three kinases, **PMK-1**, **PMK-2**, and **PMK-3**, which we call p38 map kinases due to their high sequence identity with p38. The three genes are contiguous on chromosome IV and comprise an operon. By use of a GFP reporter, we found that the promoter of the **pmks** is active throughout the intestine. An active form of **MAPK/ERK** kinase 6 (**MEK6**) phosphorylated and activated recombinant **PMK-1** and **PMK-2** in vitro. **PMK-1** and **PMK-2** phosphorylated activating transcription factor-2 (**ATF-2**), indicating an activity similar to mammalian p38. When transfected into mammalian cells, these kinases, like p38, are stimulated by osmotic stresses.
Copyright 2001 Academic Press.

L20 ANSWER 24 OF 34 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN

ACCESSION NUMBER: 1999:283047 SCISEARCH

THE GENUINE ARTICLE: 184CL

TITLE: Organization and regulation of mitogen-activated protein kinase signaling pathways

AUTHOR: Garrington T P (Reprint); Johnson G L

CORPORATE SOURCE: NATL JEWISH MED & RES CTR, DIV BASIC SCI, PROGRAM MOL SIGNAL TRANSDUCT, 1400 JACKSON ST, DENVER, CO 80206 (Reprint); CHILDRENS HOSP, DEPT PEDIATR HEMATOL ONCOL, DENVER, CO 80218

COUNTRY OF AUTHOR: USA

SOURCE: CURRENT OPINION IN CELL BIOLOGY, (APR 1999) Vol. *Adonis*

11, No. 2, pp. 211-218.

Publisher: CURRENT BIOLOGY LTD, 34-42 CLEVELAND STREET, LONDON W1P 6LE, ENGLAND.

ISSN: 0955-0674.

DOCUMENT TYPE: General Review; Journal

FILE SEGMENT: LIFE

LANGUAGE: English

REFERENCE COUNT: 58

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Mitogen-activated protein kinases (MAPKs) are components of a three kinase regulatory cascade. There are multiple members of each component family of kinases in the **MAPK** module. Specificity of regulation is achieved by organization of **MAPK** modules, in part, by use of scaffolding and anchoring proteins. Scaffold proteins bring together specific kinases for selective activation, sequestration and localization of signaling complexes. The recent elucidation of scaffolding mechanisms for **MAPK** pathways has begun to solve the puzzle of how specificity in signaling can be achieved for each **MAPK** pathway in different cell types and in response to different stimuli. As new **MAPK** members are defined, determining their organization in kinase modules will be critical in understanding their select role in cellular regulation.

L9 ANSWER 2 OF 5 MEDLINE on STN DUPLICATE 1
 ACCESSION NUMBER: 2003020540 IN-PROCESS
 DOCUMENT NUMBER: 22414944 PubMed ID: 12526744
 TITLE: Caenorhabditis elegans Innate Immune Response Triggered by Salmonella enterica Requires Intact LPS and Is Mediated by a MAPK Signaling Pathway.
 AUTHOR: Aballay Alejandro; Drenkard Eliana; Hilbun Layla R; Ausubel Frederick M
 CORPORATE SOURCE: Department of Genetics, Harvard Medical School and Department of Molecular Biology, Massachusetts General Hospital, 02114, Boston, MA, USA.
 SOURCE: CURRENT BIOLOGY, (2003 Jan 8) 13 (1) 47-52. Journal code: 9107782. ISSN: 0960-9822. *oNo*
 PUB. COUNTRY: England: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
 ENTRY DATE: Entered STN: 20030116
 Last Updated on STN: 20030116

AB Compared to mammals, insects, and plants, relatively little is known about innate immune responses in the nematode *Caenorhabditis elegans*. Previous work showed that *Salmonella enterica* serovars cause a persistent infection in the *C. elegans* intestine that triggers gonadal programmed cell death (PCD) and that *C. elegans* cell death (*ced*) mutants are more susceptible to *Salmonella*-mediated killing. To further dissect the role of PCD in *C. elegans* innate immunity, we identified both *C. elegans* and *S. enterica* factors that affect the elicitation of *Salmonella*-induced PCD. *Salmonella*-elicited PCD was shown to require the *C. elegans* homolog of the mammalian p38 mitogen-activated protein kinase (MAPK) encoded by the *pmk-1* gene. Inactivation of *pmk-1* by RNAi blocked *Salmonella*-elicited PCD, and epistasis analysis showed that *CED-9* lies downstream of *PMK-1*. Wild-type *Salmonella* lipopolysaccharide (LPS) was also shown to be required for the elicitation of PCD, as well as for persistence of *Salmonella* in the *C. elegans* intestine. However, a presumptive *C. elegans* TOLL signaling pathway did not appear to be required for the PCD response to *Salmonella*. These results establish a *PMK-1*-dependant PCD pathway as a *C. elegans* innate immune response to *Salmonella*.